DECLARATION OF PHILIP J. LANDRIGAN, M.D., M.SC. IN SUPPORT OF PETITION TO SUSPEND AND CANCEL CHLORPYRIFOS USES

I, Philip J. Landrigan, M.D., M.Sc., hereby declare and state as follows:

1. I submit this declaration in support of the petition to cancel and suspend chlorpyrifos uses that is being submitted by Earthjustice on behalf of United Farm Workers and other farmworker advocates.

PROFESSIONAL BACKGROUND AND EXPERTISE

2. I am a pediatrician and epidemiologist, and I am board certified in occupational medicine, general preventive medicine and pediatrics. I have been a member of the faculty of the Icahn School of Medicine at Mount Sinai since 1985 and am currently a professor of preventive medicine and a professor of pediatrics. I am also the Dean for Global Health, a position I have held since 2010.

3. I obtained my medical degree from Harvard Medical School in 1967. I completed an internship at Cleveland Metropolitan General Hospital and a residency in pediatrics at Boston Children's Hospital. In 1977, I received a Diploma of Industrial Health from the University of London and a Master of Science degree in Occupational Medicine from the London School of Hygiene and Tropical Medicine. My CV is attached as Exhibit 1.

4. I served for 15 years as an Epidemic Intelligence Service Officer and medical epidemiologist at the Centers for Disease Control and Prevention (CDC) and the National Institute for Occupational Safety and Health (NIOSH). I directed the national program in occupational epidemiology for NIOSH from 1979-1985. I have been awarded numerous honors throughout my career, including the Meritorious Service Medal of the U.S. Public Health Service in 1985.

5. From 2000 to 2002, I served on the Armed Forces Epidemiological Board, and from 1996 to 2005, in the Medical Corps of the U.S. Naval Reserve. I retired from the United States Navy in 2005 at the rank of Captain (O-6) and continue to serve as Surgeon General of the New York Naval Militia, the naval component of the New York National Guard.

6. I was elected a member of the Institute of Medicine of the National Academy of Sciences in 1987. I have chaired committees at the National Academy of Sciences (NAS) on Environmental Neurotoxicology and on Pesticides in the Diets of Infants and Children. From 1997 to 1998, I served as Senior Advisor on Children's Health to the Administrator of the U.S. Environmental Protection Agency (EPA) and was instrumental in helping to establish a new Office of Children's Health Protection at EPA.

7. I am editor in chief of the *Annals of Global Health*, deputy editor of the *American Journal of Industrial Medicine*, and an associate editor of *Environmental Health Perspectives.*

8. I have studied the impacts of toxic chemicals, including pesticides, on children's health for over thirty years. I have published more than 500 scientific papers and five books, on subjects including epidemiology, occupational health, environmental neurotoxicity, and children's health. I have extensive knowledge and expertise in environmental and occupational medicine, epidemiology, environmental neurotoxicity, and the effects of pesticides and other chemicals on children through my education, training, professional experience, involvement in applicable peer-reviewed research, and my ongoing review of the pertinent medical and scientific literature.

CHILDREN'S VULNERABILITY TO PESTICIDES

9. A key policy breakthrough occurred over the past three decades with the discovery that children are far more sensitive than adults to toxic chemicals in the environment.

This finding led to the recognition that chemical exposures early in life are significant yet preventable causes of disease in children and adults.

10. In the 1970s, my research showed that 60% of children living within one mile of ASARCO's El Paso smelting plant had elevated blood lead levels and that even small amounts of lead exposure lowered a child's IQ. My research showed that lead can cause brain damage to children at levels too low to clinically detect signs and symptoms. This phenomenon is now called "subclinical toxicity." These studies contributed importantly to the U.S. federal government's decision to phase out lead components from gasoline and regulate the lead content of paint in the 1970s.

11. I led a five-year study as chair of the NAS Committee that published *Pesticides in the Diets of Infants and Children* in 1993.[1] This pivotal study showed that infants and children, including infants in the womb, are much more sensitive to pesticides and other toxic chemicals than adults and documented four differences between children and adults that contribute to children's heightened susceptibility to chemicals in the environment. The following description of this work is taken from an article that I co-authored with Dr. Lynn R. Goldman, "Children's Vulnerability to Toxic Chemicals: A Challenge and Opportunity to Strengthen Health and Environmental Policy," *Health Affairs* 30, no.5 (2011): 842-850 (Exhibit 2):

> First, children have greater exposures to toxic chemicals for their body weight than adults. A six-month-old infant drinks seven times more water per pound than an adult, and children take in three to four times more calories per pound than adults. The air intake per pound of an infant is twice that of an adult. These differences result in children being disproportionately exposed to toxic chemicals in air, food, and water. Children's hand-to-mouth behavior and play on the ground further magnify their exposures.

[1] National Research Council. Pesticides in the Diets of Infants and Children. Washington, DC: National Academy Press, 1993.

> Second, children's metabolic pathways are immature, and a child's ability to metabolize toxic chemicals is different from an adult's. In some instances, infants are at lower risk than adults because they cannot convert chemicals to their toxic forms. More commonly, however, children are more vulnerable because they lack the enzymes needed to break down and remove toxic chemicals from the body.
>
> Third, children's early developmental processes are easily disrupted. Rapid, complex, and highly choreographed development takes place in prenatal life and in the first years after birth, continuing more slowly throughout childhood into puberty. In the brain, for example, billions of cells must form, move to their assigned positions, and establish trillions of precise interconnections. Likewise, development of the reproductive organs is guided by a complex and precisely timed sequence of chemical messages and is shaped by maternal and fetal hormones.
>
> Recent research in pediatrics and developmental toxicology has elaborated the concept of "windows of vulnerability." These are critical periods in early development when exposures to even minute doses of toxic chemicals—levels that would have no adverse effect on an adult—can disrupt organ formation and cause lifelong functional impairments. . . .These windows of vulnerability have no equivalent in adult life.
>
> Fourth, children have more time than adults to develop chronic diseases. Many diseases triggered by toxic chemicals, such as cancer and neurodegenerative diseases, are now understood to evolve through multistage, multiyear processes that may be initiated by exposures in infancy.

12. Since the 1993 publication of the NAS report, peer-reviewed research continues to document the developing human brain's unique vulnerability to toxic chemical exposures, and to confirm that major windows of developmental vulnerability occur *in utero,* during infancy, and in early childhood. During these sensitive life stages, exposure to pesticides and other chemicals can cause permanent brain injury at levels of exposure far below those which would have an effect in adults.

13. A fetus in the womb is at risk of exposure to pesticides and other toxic chemicals because of both exposure and vulnerabilities. In terms of exposure, the placenta does not block the passage of many toxic chemicals from the maternal to the fetal circulation. In fact, more than 200 chemicals have been detected in infants' umbilical cord blood, meaning they have passed from the mother's circulation to the baby's circulation prior to birth. In terms of susceptibility,

several prenatal developmental processes have been shown to enhance the vulnerability of the fetus in the womb to toxic chemicals.

14. Prior to the publication of the NAS report, virtually all environmental policy in the United States had focused on assessment of risk to the average adult man weighing 150 pounds. Little attention was paid to the unique risks faced by infants, children, or other vulnerable groups within the population.

15. The core findings and recommendations of the NAS report were incorporated into the 1996 Food Quality Protection Act (FQPA), which revamped federal pesticide laws. The FQPA changed risk assessment by requiring the use of child-protective safety factors to account for children's exposures and unique susceptibilities and to account also for gaps in data, and by requiring consideration of aggregate exposures to a pesticide via multiple routes, including diet, drinking water, and interaction with pesticide residues through play and other activities. It also required evaluation of cumulative effects of multiple pesticides that have the same mechanism of toxicity.

16. Implementation of the new standards led to bans on residential applications of two very widely used organophosphate insecticides: chlorpyrifos and diazinon. These bans were triggered by recognition of these compounds' neurodevelopmental toxicity to children and documentation of their long residence time in indoor environments. FQPA implementation also led to a cumulative risk assessment for all organophosphates because they have a common mechanism of toxicity, as discussed below.

NEURODEVELOPMENTAL HARM TO CHILDREN'S BRAINS FROM CHLORPYRIFOS AND OTHER ORGANOPHOSPHATES

17. Chlorpyrifos, like other organophosphate pesticides (OPs), causes acute poisonings by inhibiting the enzyme acetylcholinesterase (AChE), which regulates nerve

impulses. When cholinesterase is inhibited, it leads rapidly to overt symptoms of cholinergic hyperstimulation. The symptoms include nausea, headaches, skin rashes, eye irritation, vomiting, dizziness, seizures, coma, and death, depending on the dose and the toxicity of the product. When EPA conducted risk assessments on the organophosphates in the 1990s through 2006, it set human exposure limits based on detection of AChE inhibition. Specifically, it uses 10% red-blood cell AChE inhibition as its regulatory endpoint, called its point of departure.

18. A growing body of scientific evidence has documented neurodevelopmental harm to the developing brain from organophosphates, including chlorpyrifos. This evidence comes both from animal and epidemiology studies. EPA has compiled and reviewed the published studies in its Revised Human Health Risk Assessment for Chlorpyrifos Registration Review (Dec. 29, 2014) (RHHRA), and in its Literature Review on Neurodevelopmental Effects & FQPA Safety Factor Determination for the Organophosphate Pesticides (Sept. 15, 2015).

19. Numerous scientific studies have documented neurodevelopmental harm from prenatal and early postnatal exposures to chlorpyrifos. Animal studies have found disruption in neuronal development, neurotransmitter systems and synaptic function, as well as behavioral and cognitive impairments following low-dose perinatal chlorpyrifos exposure. Neurobehavioral effects include impairment on maze performance, locomotion, and balance in neonates exposed *in utero* or during postnatal life.

20. Direct evidence that chlorpyrifos can cause neurodevelopmental harm to children's brains comes from three epidemiology studies conducted respectively at Columbia University, University of California-Berkeley, and Mount Sinai School of Medicine. These universities conducted this research through their Centers for Children's Environmental Health and Disease Prevention Research.

21. These Centers are part of an NIH-funded, competitively awarded national network of such Centers established to increase scientific understanding of the impacts of toxic exposures on children. The Berkeley study studied children of farmworkers in the Salinas Valley of California, the Mount Sinai study observed a New York City Hispanic population whose exposures were primarily residential, and the Columbia study examined African-American and Dominican children in New York City, whose exposures were similarly residential.

22. These three Centers have been conducting long-term birth-cohort studies in which pregnant women are enrolled during their pregnancies. Their environmental exposures during pregnancy are recorded through objective measures like blood and urine samples, dust and air samples, and cord blood. Chlorpyrifos exposure during pregnancy was measured through analysis of chlorpyrifos' metabolic breakdown products in maternal urine samples. Even though these three studies were conducted in distinct geographic regions of the country, on different populations, with different routes of exposure, and using different biomarkers, they produced strongly convergent results. All studies found cognitive impairments that persist into school years from OP exposures. The Columbia study was specific to chlorpyrifos. It found that prenatal exposure to chlorpyrifos resulted in the birth of babies with reduced head circumference. Reduction in head circumference at birth is a measure of delayed or reduced brain growth during pregnancy and is an effect seen also in infants exposed in the womb to Zika virus. In the Columbia study, the degree of reduction in head circumference was proportional to the degree of maternal exposure to chlorpyrifos during pregnancy. The impact of chlorpyrifos on head circumference was no longer observed after the ban on residential application of chlorpyrifos was imposed.

23. Follow-up studies of the babies in these three studies have found that prenatal exposures have persistent deleterious effects on cognitive function through 7 years of age. The brain impairments observed in these infants and children include reduction in motor function, decreases in working and visual memory, processing speed, verbal comprehension, perceptual reasoning, and diminished IQ. The studies also documented neurobehavioral problems, including increased risk of attention deficit hyperactivity disorder, pervasive developmental disorder, and behaviors typical of the autism spectrum. Certain subpopulations demonstrate greater susceptibility, including children of farmworkers and those who have reduced capacity to detoxify OPs. Some studies found elevated risks of respiratory symptoms consistent with asthma. And recently, a study using magnetic resonance imaging found that even low to moderate levels of prenatal exposure to chlorpyrifos may lead to long-term, potentially irreversible changes in the structure of the developing brain, causing thinning of the cerebral cortex.

24. These studies found damage to children's brains from exposures to chlorpyrifos that produced no or less than 1% red-blood cell cholinesterase inhibition. In other words, the harm to the developing brain and nervous systems occurred at exposures substantially below EPA's regulatory limit, which is based on exposures that are high enough to inhibit cholinesterase in adults. EPA acknowledged in its 2014 revised human health risk assessment on chlorpyrifos that the neurodevelopmental harm to children's brains occurred at lower doses than its regulatory endpoint.

EPA'S RISK ASSESSMENTS DO NOT PROTECT AGAINST BRAIN DAMAGE TO CHILDREN

25. Even though EPA has acknowledged that neurodevelopmental harm to children occurs at exposures that produce no or only minimal cholinesterase inhibition, EPA has

continued to set its exposure limits based on cholinesterase inhibition. It continues to use 10% red-blood cell cholinesterase inhibition as the endpoint in its risk assessments, even though the mothers in the Columbia study who gave birth to infants with brain injury exhibited less than 1% cholinesterase inhibition or no inhibition at all.

26. Safety factors are used in risk assessment and standard-setting to account for uncertainties. In setting a standard or tolerance for a pesticide, EPA will begin the risk assessment by identifying an exposure level that produces no adverse effect as its endpoint. This is called the no observable adverse effect level. If some adverse effects are observed at that exposure level, EPA will add a three-fold safety factor. EPA then typically uses a tenfold safety factor to account for uncertainties in extrapolating from animal studies to people, and a second tenfold safety factor to account for differences among human populations due to such factors as genetic predisposition and other stressors. Finally, the FQPA requires EPA to use a third tenfold "child-protective" safety factor when there is either evidence that children are especially vulnerable to a chemical or when there are gaps in data concerning children's exposures or vulnerabilities. For OPs, EPA has retained a 10X child-protective FQPA safety factor because of the published evidence that these chemicals cause neurodevelopmental harm to infants and children.

27. For chlorpyrifos, however, EPA departed from this usual practice and instead relied on the Dow Agrosciences Company's pharmacokinetic-pharmacodynamic (PBPK) model of OP toxicity, which tries to pinpoint the exposures that will produce 10% cholinesterase inhibition. The Dow model is drawn largely from human studies that included deliberate dosing of people. Many of these studies were conducted in countries outside of the United States. Use of human studies in risk assessment poses significant ethical and scientific issues, and the Dow

human studies have been criticized for not meeting the informed consent standards that would be required in the US and also for scientific deficiencies. Because the Dow model uses human data, it obviates the need to extrapolate data from animals to humans. In relying on the Dow data, EPA therefore dispensed with the 10X inter-species safety factor for all populations except for women of child-bearing years. For women of child-bearing years, EPA retained the 10X intra-species safety factor because Dow did not have human data for this population.

EPA'S RISK ASSESSMENTS DO NOT PROTECT WORKERS OCUPATIONALLY EXPOSED TO CHLORPYRIFOS AND DO NOT PROTECT THE CHILDREN IN THE WOMB OF PREGNANT WOMEN WORKERS

28. In their assessments of risk from occupational exposures to chlorpyrifos, EPA identified risks of concern for over half of the handler exposure scenarios. EPA states that additional engineering controls or protective gear could eliminate the risks of concern for 27 of these activities, but notes that 126 would remain of concern regardless of the level of personal protective equipment or environmental controls. EPA also found that protection of agricultural field workers against chlorpyrifos toxicity would need longer re-entry intervals to reduce risks.

29. For many of the handler exposure scenarios, EPA found Margins of Exposure (MOEs) of less than 10 and for some scenarios the MOEs were close to or even less than 1. In other words, EPA estimates that worker exposures from these activities likely would result in 10% cholinesterase inhibition. In these scenarios, the current EPA standard manifestly fails to protect worker health or to comply with the fundamental intent of the Occupational Safety & Health Act of 1970 (OSHA) which states that every worker has the "right to a safe and healthful workplace."

30. EPA has acknowledged that its regulatory end point is underprotective. It has proposed using umbilical cord blood chlorpyrifos levels from the Columbia study to develop a more protective end point based on loss of working memory. It convened a Scientific Advisory

Panel (SAP) to review this proposal. The SAP did not support developing a point of departure based on a single study, but it did agree that EPA's approach of using 10% cholinesterase inhibition as the regulatory endpoint was underprotective.

31. The California Department of Pesticide Regulation (DPR) prepared its own risk assessment of chlorpyrifos which was modeled on EPA's approach and like EPA incorporated 10% cholinesterase inhibition, Dow's PBPK model, and the reduced safety factors. California's Office of Environmental Health Hazard Assessment (OEHHA), which routinely reviews pesticide standards proposed by DPR to ensure that they protect worker health, conducted a scientific peer review of DPR's human health risk assessments on chlorpyrifos and released its review in June 2016. OEHHA found that the 10% cholinesterase inhibition end point and the reduced safety factors proposed by the DPR failed to adequately protect human health and therefore failed to comply with occupational safety and health legislation. OEHHA recommended using a total uncertainty factor of 1000X or 3000X to protect the health of workers occupationally exposed to chlorpyrifos.

32. Any occupational exposure standard for chlorpyrifos needs to take cognizance of the fact that the workforce may include pregnant women workers (who may not yet realize that they are pregnant) and that pregnant women workers who are occupationally exposed to chlorpyrifos will unwittingly pass any chlorpyrifos that they absorb into the bodies of their unborn children where the chlorpyrifos will cause irreversible brain damage. To prevent this sequence of events, EPA should at a minimum use safety factors that total 1000X. Moreover, an additional 3X uncertainty factor is warranted over and above the 1000X safety factor because 10% cholinesterase inhibition cannot be considered a "no observable adverse effect level" in

light of the finding that neurodevelopmental harm to the fetus can result at exposure levels below this outdated limit value.

PREVENTING BRAIN DAMAGE TO CHILDREN FROM TOXIC CHEMICAL EXPOSURES YIELDS SIGNIFICANT COST SAVINGS

33. Neurobehavioral development disorders affect 10-15% of births in the United States, and the prevalence of attention deficit hyperactivity disorder, autism and other neurodevelopmental disorders is increasing in the US and worldwide. Subclinical decrements in brain function are even more common. All of these disabilities can have serious consequences for individuals, such as diminished quality of life, reduced academic achievement, behavioral disruptions, and they also have consequences for society in the form of the diminished economic productivity of affected children and the increased risk that these children will grow up to become unemployed, underemployed and institutionalized or incarcerated adults. Environmental exposures play a role in many, if not most, of these developmental disorders as genetic factors account for only approximately 30-40% of them.

34. Preventing exposures to chemicals can yield great economic savings. While it is difficult to precisely quantify the harm from neurodevelopmental disorders and the cost savings that result from their prevention, several studies suggest that both are quite large. To estimate the contribution of environmental pollutants to the prevalence and costs of disease in American children, investigators at Mount Sinai School of Medicine examined four categories of illness: lead poisoning, asthma, cancer, and neurobehavioral disorders. Based on prevalence, the environmentally attributable fraction of each disease, and national economic data, they calculated that the total annual costs of these diseases attributable to environmental exposures is $54.9 billion (range $48.8 billion to $64.8 billion): $43.4 billion for lead poisoning, $2.0 billion for asthma, $0.3 billion for childhood cancer, and $9.2 billion for neurobehavioral disorders.

Because of the difficulties inherent in assessing the full economic consequences of neurobehavioral impairments, it is likely that these estimates are low.

35. After the phase-out of lead in gasoline from 1976 and 1990, the mean blood lead level of American children decreased by more than 90% (to below 2 micrograms per deciliter today), and the incidence of childhood lead poisoning also fell by more than 90%. A further consequence of the reduction in exposure to lead was that the mean IQ of American children has increased. Children born in the United States today are estimated to have IQ scores that, on average, are 2.2–4.7 points higher than those of children born in the early 1970s. And because each 1-point gain in population mean IQ is associated with an estimated 2% increase in productivity over a lifetime, the gain in population IQ is estimated to have produced a national economic benefit of $110–$319 billion in each annual cohort of babies born in the United States since the 1980s.

36. Dr. David Bellinger, a professor of neurology at Harvard Medical School, published a paper in 2012, which estimated that Americans have collectively forfeited 41 million IQ points as a result of exposure to lead, mercury, and OPs. He calculated a total loss of 16.9 million IQ points due to exposure to OPs.[2]

EPA'S APPROACH TO WORKER RISK MITIGATION IS UNDERPROTECTIVE AND AT ODDS WITH STANDARD OCCUPATIONAL HEALTH PRACTICE

37. When EPA identifies a risk of concern, it explores as a first priority whether use of personal protective equipment will eliminate the risk. If personal protective equipment is found not to be protective, EPA then asks whether engineering controls or administrative controls such as restricted re-entry intervals will eliminate the risk. Only if the risk of concern

[2] D.C. Bellinger, "A Strategy for Comparing Contributions of Environmental Chemicals and Other Risk Factors to Neurodevelopment of Children," *Environmental Health Perspectives*, Vol. 120, No. 4, pp.501-507 (April 2012).

remains after implementation of all such mitigation does EPA explore eliminating the exposure or shifting to less harmful alternative chemicals or application methods.

38. EPA's approach is backwards and wrong. It violates standard, long-established occupational health practice. It fails to protect worker health.

39. In the field of occupational safety and health, regulators adhere to a hierarchy of controls that prioritizes prevention of exposure – not use of personal protection. Regulators start by asking whether the exposure can be eliminated altogether or whether other less toxic chemicals can be substituted. If those approaches are found not to be feasible, the regulator will look to engineering controls such as machine-guarding or administrative controls such as longer re-entry times to sprayed fields. The regulator will turn to personal protective equipment only as a last resort, because personal protective equipment has been shown repeatedly over the decades to be far less effective at worker protection than product substitution, engineering controls and administrative controls. A final reason for not relying on personal protective equipment is that such equipment degrades workers' ability to function and increases risk of heat stress and heat stroke. Thus double-layers of clothing, gloves, and respirators likely impede mobility and contribute to heat and respiratory stress of pesticide handlers working in hot temperatures during summer growing seasons.

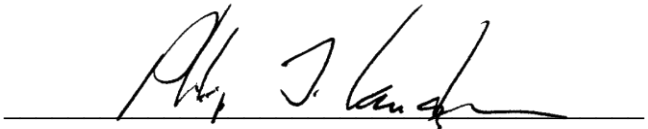
40. OSHA has adhered to this prioritization for decades. The lead standard is illustrative. EPA refused to rely on personal protective equipment, and on respirators in particular, because they fail to eliminate exposure, provides inadequate protection, and creates additional hazards by interfering with vision and mobility. The 1978 lead standard is replete with findings that respirators afford inadequate protection. OSHA required respirators **in**

addition to engineering controls to afford workers additional protection during the time it would take to fully implement the controls. 43 Fed. Reg. 52,952 (Nov. 14, 1978).

41. For decades, EPA has adopted a wrong-headed strategy for mitigating worker exposures to chlorpyrifos and other toxic pesticides that relies first and foremost on personal protective equipment. By relying on this inadequate strategy and by relying on personal protective equipment that has been shown to confer highly inadequate protection, EPA has allowed workers to be exposed to harmful levels of chlorpyrifos. By relying on this ineffective strategy, EPA has allowed pregnant women workers to be occupationally exposed to levels of chlorpyrifos that can result in fetal brain damage to infants in the womb. Sound occupational health principles require engineering or administrative controls, where effective, or elimination of the exposure, where engineering or administrative controls are not effective.

I declare under penalty of perjury that the foregoing is true and correct.

Executed on this 7th day of September 2016, in New York, New York.



Philip J. Landrigan, M.D., M.Sc.

Exhibit 1

CURRICULUM VITAE

Philip J. Landrigan, M.D., M.Sc., D.I.H., F.A.A.P., F.A.C.P.M.

ACADEMIC APPOINTMENTS

Current:

Icahn School of Medicine at Mount Sinai, Dean for Global Health, 2010-Present

Icahn School of Medicine at Mount Sinai, Professor, Department of Preventive Medicine, 1990-Present

Icahn School of Medicine at Mount Sinai, Professor of Pediatrics, 1985-Present

Previous:

Icahn School of Medicine at Mount Sinai, Ethel H. Wise Professor and Chairman, Department of Preventive Medicine, 1990-2015.

Icahn School of Medicine at Mount Sinai, Director, Division of Environmental and Occupational Medicine, Department of Community and Preventive Medicine, 1985-1990.

U.S. Environmental Protection Agency, Senior Advisor to the Administrator on Children's Health and the Environment, 1997-1998. (Sabbatical position)

National Institute for Occupational Safety and Health, Director, Division of Surveillance, Hazard Evaluations and Field Studies, 1979-1985.

Centers for Disease Control and Prevention

- Chief, Environmental Hazards Activity, Bureau of Epidemiology, 1974-1979.
- Director, Research and Development, Bureau of Smallpox Eradication, 1973-1974.
- Epidemic Intelligence Service (EIS) Officer, 1970-1973.

Adjunct Positions:

Harvard School of Public Health, Adjunct Professor of Environmental Health, 2010-present; Visiting Lecturer on Occupational Health, 1981-2010

Harvard Medical School, Clinical Instructor in Pediatrics, 1969-1970; Visiting Lecturer on Preventive Medicine and Clinical Epidemiology, 1982-Present

University of Washington School of Public Health and Community Medicine, Auxilary Clinical Professor of Environmental Health, 1983-2013

University of Cincinnati, Department of Environmental Health, College of Medicine, Assistant Clinical Professor of Environmental Health, 1981-1986

London School of Hygiene and Tropical Medicine, Visiting Fellow, TUC Institute of Occupational Health, 1976-1977

EDUCATION

High School: Boston Latin School, 1959
College: Boston College, A.B. (magna cum laude), 1963
Medical School: Harvard Medical School, M.D., 1967

POSTDOCTORAL TRAINING

Internship: Cleveland Metropolitan General Hospital, 1967-1968
Residency: Children's Hospital Medical Center, Boston, (Pediatrics), 1968-1970
Post Graduate: London School of Hygiene & Tropical Medicine, 1976-77
Diploma of Industrial Health (England), 1977
Master of Science in Occupational Medicine, University of London (with distinction), 1977

CERTIFICATION

American Board of Pediatrics - 1973
American Board of Preventive Medicine:
General Preventive Medicine - l979
Occupational Medicine - 1983

MEDICAL LICENSURE

Massachusetts #31277, 1967 - present
New York #162034, 1985 - present

INSTITUTE OF MEDICINE

Institute of Medicine, National Academy of Sciences, Elected to membership, 1987

HONORS/AWARDS

Asbestos Disease Awareness Association – Dr. Irving Selikoff Lifetime Achievement Award, 2016
Grassroots Environmental Education, Award for Outstanding Leadership in Children's Environmental Health, 2015
Boston College – Distinguished Alumni Research Award, 2014
Boston Latin School - Distinguished Graduate Award, 2014
University of Medicine & Dentistry of New Jersey - Senator Frank R. Lautenberg Annual Award in Public Health, 2011
Hearst Foundation, The Daily Green – The Heart of Green Award, 2010
The New York Academy of Medicine – The Stephen Smith Medal for Lifetime Achievement in Public Health, 2009
U.S. Environmental Protection Agency, Region II – Environmental Quality Award on behalf of Mount Sinai Medical Center, 2009
Westchester County (NY). Sustainability Award for Service on Westchester County Global Warming Task Force, 2009
Student Physicians for Social Responsibility. Lifetime Achievement Award, 2009
Women's City Club of New York. Civic Spirit Award, 2009
Boston College. Alumni Award for Professional Excellence, 2008
Collegium Ramazzini. Irving J. Selikoff Award, 2008
Healthy Schools Network, Inc. Healthy Schools Hero Award, 2008
Westchester Children's Association. Edith Macy Award for Distinguished Service, 2008
Children's Health Environmental Coalition. Lifetime Achievement Award, 2006
U.S. Environmental Protection Agency. Child Health Champion Award, 2006
Huntington Breast Cancer Action Coalition. Humanities Award for Children's Health Protection, 2005
Icahn School of Medicine at Mount Sinai. J. Lester Gabrilove Award, 2005
American College of Occupational and Environmental Medicine. Health Achievement in Occupational Medicine Award, 2005
National Nutritional Foods Association. Rachel Carson Environmental Award, 2005
Federated Conservationists of Westchester County. Super Hero Award for Children's Health, 2005
Physicians for Social Responsibility, Los Angeles Chapter. Socially Responsible Medicine Award, 2004
***Organic Style Magazine*.** Environmental Power List, 2004
Finnish Institute for Occupational Health. Jorma Rantanen Award, 2003
American Public Health Association, David P. Rall Award for Advocacy in Public Health, 2003
Castle Connolly Ltd.-America's Top Doctor. Preventive Medicine. New York Metropolitan Area and United States 2001, 2002, 2003, 2004, 2005, 2006, 2007, 2008, 2009, 2010, 2011, 2012, 2013, 2014 and 2015

HONORS/AWARDS (cont)

Public Health Association of New York City, Haven Emerson Award, 2002
National Institute for Occupational Safety & Health, James Keogh Award, 2002
Icahn School of Medicine at Mount Sinai, Jacobi Medallion, 2002
Environmental Advocates (New York), Award for Environmental Advocacy on Behalf of Children, 2000
American Conference of Governmental Industrial Hygienists, William Steiger Memorial Award, 2000
Russian Academy of Medical Science, Elected as Foreign Member, 2000
Earth Day New York, Award for Excellence in Environmental Medicine, 1999
Mothers & Others for a Livable Planet, Award for Advocacy on Behalf of the Health of Children, 1999
American College of Preventive Medicine, Katherine Boucot Sturgis Award, 1999
International Society for Occupational and Environmental Health, Vernon Houk Award, 1998
New Jersey Environmental Federation Certificate of Recognition. Environmental Achievement Award, 1998
Physicians for Social Responsibility, Broad Street Pump Award in Environmental Health, 1996
International Association of Fire Fighters, Occupational Health and Safety Award, 1995
American Public Health Association, Herbert L. Needleman Medal and Award for Scientific Contributions and Advocacy on Behalf of Children, 1995
United Brotherhood of Carpenters, William Sidell Presidential Award, 1995
New England College of Occupational and Environmental Medicine, Harriet Hardy Award, 1993
New York Committee for Occupational Safety and Health, Annual Honoree, 1985
United States Navy

- Navy & Marine Corps Commendation Medal (3 awards), 2002, 2003 and 2005
- National Defense Service Medal, 2003
- Secretary of Defense Medal for Outstanding Public Service, 2002

U.S. Public Health Service

- Meritorious Service Medal, 1985
- Group Citation as Member of Beryllium Review Panel, 1978
- Career Development Award, 1976

U.S. Department of Health, Education and Welfare, Volunteer Award, 1973

HONORARY DEGREES:

Mount Sinai School of Medicine, Doctor of Science (honoris causa), 2007

OTHER PROFESSIONAL APPOINTMENTS:

American College of Preventive Medicine Fellow, 2003-present
Physicians for Social Responsibility, Board of Directors 1996-1999; Board of Sponsors, 1994-95
New York Academy of Medicine, Elected Fellow, 1991
American College of Occupational and Environmental Medicine, Fellow, 1986
Herman Biggs Society, Member, 1986-1992
International Commission on Occupational Health, Member, 1985-present
Collegium Ramazzini, Fellow, 1983-present
President, 1997-present
American College of Epidemiology, Fellow, 1983-present
Board of Directors, 1990 - 1993
American Epidemiological Society, Elected Member, 1982-present
American Public Health Association, Member, 1982-present
Occupational Health Section, Chair, 1989-90

OTHER PROFESSIONAL APPOINTMENTS: (cont)

Society for Epidemiologic Research, Member, 1978-present
Royal Society of Medicine, Elected Fellow, 1977
American Academy of Pediatrics, Fellow, 1975-present
New York Occupational Medicine Association, Member 1985-present
Board of Directors, 1988-1990
New York Academy of Sciences, Fellow 2002-present

COMMITTEES:

The White House

Presidential Advisory Committee on Gulf War Veterans' Illnesses, 1995-1996

American Academy of Pediatrics

Committee on Environmental Hazards, 1976-1987. Chairman, 1983-1987

National Research Council

Institute of Medicine, Chairman, Interest Group (14) Environmental and Occupational Health and Toxicology, 2009-2011
National Academy of Sciences, Board on Sustainable Development, 1995-1998
National Academy of Sciences, Committee on the Scientific Issues Surrounding the Regulation of Pesticides in the Diets of Infants and Children, Chairman, 1988-1992
National Academy of Sciences, Committee on Neurotoxicology in Risk Assessment, 1987-1989
National Academy of Sciences, Committee on the Epidemiology of Air Pollutants, Vice-Chairman, 1984-1985
National Academy of Sciences, Assembly of Life Sciences, 1981-1982; Commission on Life Sciences, 1982-1984
National Academy of Sciences, Panel on the Proposed Air Force Study of Herbicide Agent Orange, 1979-1980
Institute of Medicine, Committee for a Planning Study for an Ongoing Study of Costs of Environment-Related Health Effects, 1979-1980
National Academy of Sciences, Assembly of Life Sciences. Board on Toxicology and Environmental Health Hazards, 1978-1987; Vice-Chairman, 1981-1984

National Institutes of Health/U.S. Public Health Service

National Institutes of Health, National Institute of Environmental Health Sciences, External Clinical Advisory Council, 2009-present
National Institute of Child Health and Human Development, Federal Advisory Committee to the National Children's Study, 2003-2005
National Institute of Child Health and Human Development, National Children's Study, Executive Steering Committee, 2007-2009
Food and Drug Administration, Ranch Hand Advisory Committee, 2000-2001
National Institute for Occupational Safety and Health, Board of Scientific Counselors, 1995-1997
National Institutes of Health, Study Section on Epidemiology and Disease Control, 1986-1990
National Institute of Environmental Health Sciences, Third Task Force for Research Planning in the Environmental Health Sciences; Chairman, Subtask Force on Research Strategies for Prevention of and Intervention in Environmentally Produced Disease, 1983-1984

Department of Defense

Armed Forces Epidemiological Board, 2000-2002

COMMITTEES: (cont)

State and Local Government

New York State, Governor's Advisory Committee on Safety and Healthy New York Foods, 2015-2016
State of New York, Advisory Council on Children's Environmental Health, Co-Chair 2009-present
State of New York, Advisory Council on Lead Poisoning Prevention, 2009-present
Westchester County, New York, Westchester County Global Warming Task Force, 2006-2008
City of New York, Weapons of Mass Destruction (WMD) Advisory Group, 2002-2008
State of New York, Health Research Science Board, 1997-present
State of New York, Public Health Priorities Committee, 1996
State of New York, New York State Advisory Council on Lead Poisoning Prevention, Chairman, 1993-2004
City of New York, Mayor's Lead Paint Poisoning Advisory Committee, 1991-1993
State of New York, Asbestos Advisory Board, Chair, 1987-present
State of New Jersey, Meadowlands Cancer Advisory Board, Chair, 1987-1989
State of New York, Governor's Blue Ribbon Committee on the Love Canal, 1978-1979

Academic

Cornell University, Dean's Advisory Council in Veterinary Medicine, 1996-1997
Mickey Leland National Urban Air Toxics Research Center, National Advisory Committee, 1994-1995
New York Academy of Medicine, Working Group on Housing and Health, 1987-1989; Chairman, 1989
New York Lung Association, Research and Scientific Advisory Committee, 1986-1989. Board of Directors, 1987-1990
Association of University Programs in Occupational Health and Safety, 1985-Present; President, 1986-1988
Milbank Memorial Foundation, Technical Board, 1986-1988
Harvard School of Public Health, Occupational Health Program, Residency Review Committee, 1981-1983; Chairman, 1981

International Organizations

World Health Organization. Contributor to the WHO Publication: "Guidelines on Studies in Environmental Epidemiology" (Environmental Health Criteria, No. 27), 1984.
International Agency for Research on Cancer, service as member of Working Groups on Cancer Assessment, volume 29 (benezene), 1981; volume 42 (silica), 1986; voume 87 (lead), 2005; volume 98, (firefighting) 2007; volume 100 (asbestos). 2011.

Environmental Organizations

Healthy Child, Healthy World, Board of Directors, 1996-present
Children's Environmental Health Network, Board of Directors, 1995-present
Environmental Health Foundation, Board of Directors, 1993-1996
INFORM, Board of Directors, 1991-2003

Labor Unions

International Brotherhood of Teamsters, National Health and Safety Advisory Committee, 1994-2002
George Meany Center for Labor Studies, Board of Trustees, 1994-1997
United Brotherhood of Carpenters, National Health and Safety Fund, Medical Advisory Committee, 1990 -2000; Chairman, 1994-2000

COMMITTEES: (cont)

Labor Unions

United Automobile Workers (UAW) - Chrysler Corporation, Joint Scientific Advisory Committee, Member, 1990-2006

International Association of Fire Fighters, John Redmond Foundation, Medical Advisory Committee, 1989-present

Other Organizations

Health Insurance Plan (HIP) of Greater New York, Board of Directors, 1992-1994

American Legion, Science Panel, Chairman, 1988-2000

Editorial Boards

Editor-in-Chief: *Annals of Global Health*, 2013-present

Deputy Editor: *American Journal of Industrial Medicine,* 2007-present

Associate Editor: *Environmental Health Perspectives,* 2002-present

Editorial Board: *Journal of Public Health Management and Practice*, 1995-1996

Editor-in-Chief: *American Journal of Industrial Medicine*, 1992-2006; Consulting Editor, 1979-1992

Editorial Board: *New Solutions*: A Journal of Environmental and Occupational Health Policy, 1990-present

Editorial Board: *The PSR Quarterly*, 1990-1994

Editorial Board: *American Journal of Public Health*, 1987-1993

Editor-in-Chief: *Environmental Research*, 1987-1994

Senior Editor: *Environmental Research*, 1985-1987

Editorial Board: *Annual Review of Public Health*, 1984-1990

Consulting Editor: *Archives of Environmental Health*, 1982-present

National Service

United States Naval Reserve, Medical Corps, 1996-2005
LCDR (0-4) 1996-98; CDR (0-5) 1998 – 2004; CAPT (0-6) 2004-2005. Retired January 1, 2005.

United States Public Health Service, Commissioned Corps, 1970-1995. LCDR (0-4) to CAPT (0-6).

New York Naval Militia 2000-present; CAPT (0-6); Surgeon General.

GRANT SUPPORT

ACTIVE:

Blacksmith Institute (Landrigan, PI) 01/01/12 – 12/23/17
$45,000

Assessing the Disease Burden of Hazardous Waste Sites

The purpose of this contract is to support the development of a series of scientific papers that will assess the health burden associated with human exposure to hazardous waste sites in the developing world.

2011-N-13318 (Lucchini, PI) 07/01/11 – 12/31/16
CDC $28,422,550

World Trade Center Data and Coordination Center

This project is the coordinating center for a multicenter program providing monitoring and treatment to volunteers who assisted in the recovery and cleanup after the 9/11 attack.

Role: Co-Investigator

Completed Research Support:

1T32HD049311 (Landrigan, PI) 05/01/07 - 07/01/13
NICHD $323,002
Research Training Program in Environmental Pediatrics
The goal of this interdisciplinary research training program is to train the next generation of physician-researchers and academic leaders in environmental pediatrics.

C-010124 NYS DoH Landrigan (PI) 4/1/09 – 3/31/12; Lucchini (PI) 4/1/12 – present
World Trade Center Responders Data and Coordinating Center. This program has collected, analyzed and published medical monitoring and treatment data collected clinically on 30,000 9/11 responders evaluated at five Clinical Centers in the New York metropolitan area.

NIH-HHSN27520080031C (Landrigan, PI) 09/28/08 - 09/27/13 (Monroe)
NIH
National Children's Study Vanguard Centers
This project will recruit 1250 live births into a NICHD study of social, behavioral and environmental factors and their impact on childhood health, growth and development. The Queens Vanguard Center is one of the first six sites selected to pilot the NCS, which will follow more than 100,000 children across the United States from birth until age 21.

U10-OH08232 CDC Landrigan (PI) 6/1/04 – 3/31/12; Lucchini (PI) 4/1/12 - present
New York/New Jersey Education Research Center in Occupational Safety & Health. The goal of this multi-institutional program is to train professionals from multiple disciplines - medicine, nursing, industrial hygiene and industrial safety - to be future leaders in occupational health and safety.

Children's Environmental Health Center - Inner City Toxicants, Child Growth and Development
Co-Principal Investigator
EPA RD831711-01 11/1/03 – 10/31/10
NIEHS P01 ES009584 11/1/98 – 10/31/10

ORIGINAL, PEER-REVIEWED PUBLICATIONS

1. Lovejoy FH Jr, Marcuse EK, Landrigan PJ: Two examples of purpura factitia. *Clinica Pediatr* 10:183-184, 1971.
2. Landrigan PJ, Conrad JL: Current status of measles in the United States. *J Infect Dis* 124:620-622, 1971.
3. Landrigan PJ: Epidemic measles in a divided city. *JAMA* 221:567-570, 1972.
4. Hattwick MA, Hochberg FH, Landrigan PJ, Gregg MG: Skunk-associated human rabies. *JAMA* 222:44-50, 1972.
5. Grand MG, Wyll SA, Gehlbach SH, Landrigan PJ, Judelsohn RG, Zendel SA, Witte JJ: Clinical reactions following rubella vaccination: A prospective analysis of joint, muscular, and neuritic symptoms. *JAMA* 220:1569-1572, 1972.
6. Landrigan PJ, Griesbach PH: Measles - In previously vaccinated children in Illinois. *Illinois Med J* 141:367-372, 1972.
7. Tarlin L, Landrigan PJ, Babineau R, Alpert JJ: A comparison of the antipyretic effect of acetaminophen and aspirin: Another approach to poison prevention. *Am J Dis Child* 124:880-882, 1972.
8. Landrigan PJ, Witte JJ: Neurologic disorders following live measles-virus vaccination. *JAMA* 223:1459-1462, 1973.
9. Landrigan PJ, Murphy KB, Meyer HM, Parkman PD, Eddins DL, Witte JJ: Combined measles-rubella vaccines: virus dose and serologic response. *Am J Dis Child* 125:65-67, 1973.
10. Landrigan PJ, Huber DH, Murphy GC, Creech WB, Bryan JA: The protective efficacy of immune serum globulin in hepatitis A: A statistical approach. *JAMA* 223:74-75, 1973.
11. Landrigan PJ, Bresnan M, Berenberg W: Behr's syndrome: Familial optic atrophy, spastic diplegia, and ataxia. *Develop Med Child Neurol* 15:41-47, 1973.
12. Brandling-Bennett AD, Landrigan PJ, Baker EL: Failure of vaccinated school children to transmit measles. *JAMA* 224:616-618, 1973.
13. Barthel WF, Smrek AL, Angel GP, Liddle JA, Landrigan PJ, Gehlbach SH, Chisholm JJ: Modified Delves' cup atomic absorption determination of lead in blood. *J Official Analyst Chemists* 56:1252-1256, 1973.
14. Schluederberg A, Lamm SH, Landrigan PJ, Black FL: Measles immunity in children vaccinated before one year of age. *Am J Epidemiol* 97:402-409, 1973.
15. Landrigan PJ, Stoffels MA, Anderson E, Witte JJ: Epidemic rubella in adolescent boys - clinical features and results of vaccination. *JAMA* 227:1283-1287, 1974.
16. Marshall R, Habicht JP, Landrigan PJ, Foege WH, Delgado H: Effectiveness of measles vaccine given simultaneously with DTP. *J Trop Pediatr* 20:126-129, 1974.
17. Landrigan PJ, Navarro E, Eddins D: Epidemiologic assessment of a nationwide multiple antigen vaccine campaign. *J Trop Pediatr* 20:135-140, 1974.
18. Landrigan PJ, Gehlbach SH, Rosenblum BF, Shoults JM, Candelaria RM, Barthel WF, Liddle JA, Smrek AL, Staehling NW, Sanders JF: Epidemic lead absorption near an ore smelter: the role of particulate lead. *New Engl J Med* 292:123-129, 1975.
19. Landrigan PJ, Whitworth RH, Baloh RW, Barthel WF, Staehling NW, Rosenblum BF: Neuropsychological dysfunction in children with chronic low-level lead absorption. *Lancet* 1:708-712, 1975.

ORIGINAL, PEER-REVIEWED PUBLICATIONS (cont)

20. Landrigan PJ, McKinney AS, Hopkins LC, Rhodes WW Jr, Price WA, Cox DH: Chronic lead absorption - result of poor ventilation in an outdoor pistol range. *JAMA* 234:394-397, 1975.
21. Wallace RB, Landrigan PJ, Smith EA, Pifer J, Teller B, Foster SO: Trial of a reduced dose of measles vaccine in Nigerian children. *Bull WHO* 53:361-364, 1976.
22. Landrigan PJ, Baker EL, Feldman RH, Cox DH, Eden KV, Orenstein WA, Mather JA, Yankel AJ, VonLindern IH: Increased lead absorption with anemia and slowed nerve conduction in children near a lead smelter. *J Pediatr* 85:904-910, 1976.
23. Levine RJ, Moore RM, McLaren CD, Barthel WF, Landrigan PJ: Occupational lead poisoning, animal deaths, and environmental contamination at a scrap smelter. *Am J Public Health* 66:548-552, 1976.
24. Baker EL, Folland DS, Taylor TA, Frank M, Peterson W, Lovejoy G, Cox D, Housworth J, Landrigan PJ: Lead poisoning in children of lead workers: Home contamination with industrial dust. *New Engl J Med* 296:260-261, 1977.
25. Diggory HJP, Landrigan PJ, Latimer KP, Ellington AC, Kimbrough RD, Liddle JA, Cline AE, Smrek AL: Fatal parathion poisoning caused by contamination of flour in international commerce. *Am J Epidemiol* 106:145-153, 1977.
26. Winegar DA, Levy BS, Andrews JS Jr, Landrigan PJ, Scruton WH, Krause MJ: Chronic occupational exposure to lead: an evaluation of the health of smelter workers. *J Occup Med* 19:603-606, 1977.
27. Baker EL, Smrek A, Kimbrough RD, Hudgins M, Landrigan PJ, Liddle JA: Hereditary cholinesterase deficiency: A report of a family with two rare genotypes. *Clinical Genetics* 12:134-138, 1977.
28. Baker EL Jr, Hayes CG, Landrigan PJ, Handke JL, Leger RT, Housworth WJ, Harrington JM: A nationwide survey of heavy metal absorption in children living near primary copper, lead and zinc smelters. *Am J Epidemiol* 106:261-273, 1977.
29. Baker EL, Field PH, Basteyns BJ, Skinner GH, Bertozzi PE, Landrigan PJ: Phenol poisoning due to contaminated drinking water. *Arch Environ Health* 33:89-94, 1978.
30. Wysowski DK, Landrigan PJ, Ferguson SW, Fontaine RE, Liddle JA: Cadmium exposure in a community near a smelter. *Am J Epidemiol* 107:27-35, 1978.
31. Cannon SB, Veazey JM, Jackson RS, Burse VW, Hayes C, Straub WE, Landrigan PJ: Epidemic kepone poisoning in chemical workers. *Am J Epidemiol* 107:529-537, 1978.
32. Harrington JM, Craun GF, Meigs JW, Landrigan PJ, Flannery JT, Woodhull RS: An investigation of the use of asbestos cement pipe for public water supply and the incidence of gastrointestinal cancer in Connecticut 1935-1973. *Am J Epidemiol* 107:96-103, 1978.
33. Baker EL, Peterson WA, Holtz J, Mann JM, Coleman C, Landrigan PJ: Subacute cadmium intoxication in jewelry workers: An evaluation of diagnostic procedures. *Arch Environ Health* 34:173-177, 1979.
34. Morse DL, Baker EL, Landrigan PJ: Cut flowers: A potential pesticide hazard. *Am J Public Health* 69:53-56, 1979.
35. Morse DL, Watson WN, Housworth J, Witherell LE, Landrigan, PJ: Exposure of children to lead in drinking water. *Am J Public Health* 69:711-712, 1979.
36. Morse DL, Kominsky JR, Wisseman CL III, Landrigan PJ: Occupational exposure to hexachlorocyclopentadiene: How safe is sewage? *JAMA* 241:2177-2179, 1979.

37. Morse DL, Landrigan PJ, Rosenblum BF, Housworth J: El Paso revisited: epidemiologic follow-up of an environmental lead problem. *JAMA* 242:739-741, 1979.
38. Morse DL, Harrington JM, Kelter A, Housworth J, Landrigan PJ: Arsenic exposure in multiple environmental media in children near a smelter. *Clin Toxicol* 14:389-399, 1979.
39. Landrigan PJ, Wilcox KR Jr, Silva J Jr, Humphrey HEB, Kauffman C, Heath CW Jr: Cohort study of Michigan residents exposed to polybrominated biphenyls: epidemiologic and immunologic findings. *Ann NY Acad Sci* 320:284-294, 1979.
40. Baker EL, Landrigan PJ, Barbour AG, Cox DH, Folland DS, Ligo RN, Throckmorton J: Occupational lead poisoning in the United States: Clinical and biochemical findings related to blood lead levels. *Br J Ind Med* 36:314-322, 1979.
41. Silva J, Kauffman CA, Simon DG, Landrigan PJ, Humphrey HEB, Heath CW, Wilcox ER, VanAmburg G, Kaslow RA, Hoff K: Lymphocyte function in humans exposed to polybrominated biphenyls. *J Recituloendothelial Soc* 26:341-347, 1979.
42. Englender SJ, Landrigan PJ, Atwood RB, Clarkson TW: Organic mercury exposure from fungicide-contaminated eggs. *Arch Environ Health* 35:224-228, 1980.
43. Baker EL, Landrigan PJ, Glueck CJ, Zack MM, Liddle JA, Burse VW, Housworth WF, Bayse DD, Needham LL: Metabolic consequences of exposure to polychlorinated biphenyls (PCBs) in sewage sludge. *Am J Epidemiol* 112:553-563, 1980.
44. Landrigan PJ, Tamblyn PB, Nelson M, Kerndt P, Kronoveter KJ, Zack MM: Lead exposure in stained glass workers. *Am J Ind Med* 11:177-180, 1980.
45. Wilson R, Lovejoy FH, Jaeger RJ, Landrigan PJ: Acute phosphine poisoning aboard a grain freighter: epidemiologic, clinical, and pathological findings. *JAMA* 244:148-150, 1980.
46. Rosenberg MJ, Landrigan PJ, Hahn JL, Crowley S: Low-level arsenic exposure in wood processing plants. *Am J Ind Med* 1:99-108, 1980.
47. Nelson DB, Kimbrough ED, Landrigan PJ, Hayes AW, Yang GC, Benanides J: Aflatoxin and Reye's Syndrome: A case-control study. *Pediatrics* 66:865-869, 1980.
48. Hassan A, Velasques E, Belmar R, Coye M, Drucker E, Landrigan PJ, Michaels D, Sidel KB: Mercury poisoning in Nicaragua: A case study of the export of environmental and occupational health hazards by a multinational corporation. *Int J Health Serv* 11:221-226, 1981.
49. Halperin W, Landrigan PJ, Altman R, Iaci AW, Morse DL, Needham LL: Chemical fire at toxic waste disposal plant: Epidemiologic study of exposure to smoke and fumes. *J New Jersey Med Soc* 78:592-594, 1981.
50. Rinsky RA, Zumwalde RD, Waxweiler RJ, Murray WE, Bierbaum PJ: Landrigan PJ, Terpilak M, Cox C: Cancer mortality at a naval nuclear shipyard. *Lancet* 1:231-235, 1981.
51. Froneberg B, Johnson PL, Landrigan PJ: Respiratory illness caused by overheating of polyvinyl chloride. *Br J Ind Med* 39:239-243, 1982.
52. Landrigan PJ, Baker EL, Himmelstein JS, Stein GF, Weddig JP, Straub WE: Exposure to lead from the Mystic River Bridge - The dilemma of deleading. *New Engl J Med* 306:673-676, 1982.
53. Schulte PA, Singal M, Stringer WT, Kominsky JR, Landrigan PJ: The efficacy of a population-based comparison group in cross-sectional occupational health studies. *Am J Epidemiol* 116:981-989, 1982.

54. Landrigan PJ, Costello RJ, Stringer WT: Occupational exposure to arsine: An epidemiologic reappraisal of current standards. *Scand J Work Environ Health* 8:169-177, 1982.
55. Halperin WE, Goodman M, Stayner L, Elliott LJ, Keenyside RA, Landrigan PJ: Nasal cancer in a worker exposed to formaldehyde. *JAMA* 249:510-512, 1983.
56. Landrigan PJ, Powell KE, James LM, Taylor PR: Paraquat and marijuana: epidemiologic risk assessment. *Am J Public Health* 73:784-788, 1983.
57. Kreiss K, Zack MM, Landrigan PJ, Feldman RG, Niles CA, Chirico-Post J, Sax DS, Boyd MH, Cox DH: Neurologic evaluation of a population exposed to arsenic in Alaskan well water. *Arch Environ Health* 38:116-121, 1983.
58. Landrigan PJ, Miller BP: The Arjenyattah Epidemic: Home interview data and toxicological aspects. *Lancet* 2:1474-1475, 1983.
59. Horan JM, Kurt T, Landrigan PJ, Melius JM, Singal M: Neurologic dysfunction from exposure to 2-t-butylazo-2-hydroxy-5-methylhexane (BHMH): A new occupational neuropathy. *Am J Public Health* 75:513-517, 1985.
60. Selevan SG, Landrigan PJ, Stern FB, Jones JH: Mortality of lead smelter workers. *Am J Epidemiol* 122:673-683, 1985.
61. Robins JM, Landrigan PJ, Robis TG, Fine LJ: Decision-making under uncertainty in the setting of environmental health regulations. *J Public Health Policy* 3:322-328, 1985.
62. Landrigan PJ, Straub WE: Occupational lead exposure aboard a tall ship. *Am J Ind Med* 8:233-239, 1985.
63. Landrigan PJ, Cherniack MG, Lewis FA, Catlett LR: Silicosis in a grey iron foundry: The persistence of an ancient disease. *Scand J Work Environ Health* 12:32-39, 1986.
64. Liss GM, Halperin WE, Landrigan PJ: Occupational asthma in a home pieceworker. *Arch Environ Health* 41:359-326, 1986.
65. Stern FB, Waxweiler RA, Beaumont JJ, Lee ST, Halperin WE, Zumwalde RD, Bierbaum PJ, Rinsky RA, Landrigan PJ, Murray WE: A case-control study of leukemia at a naval nuclear shipyard. *Am J Epidemiol* 123: 980-992, 1986.
66. Wielopolski K, Ellis KJ, Vaswani AN, Cohn SH, Greenberg A, Puschett JB, Parkinson DK, Fetterolf DE, Landrigan PJ: In vivo bone lead measurements: A rapid monitoring method for cumulative lead exposure. *Am J Ind Med* 9:221-226, 1986.
67. Greenberg A, Parkinson DK, Fetterolf DE, Ellis KJ, Wielopolski L, Vaswani AN, Cohn SH, Landrigan PJ, Puschett JB: Effects of elevated lead and cadmium burdens on renal function and calcium metabolism. *Arch Environ Health* 4:69-76, 1986.
68. Landrigan PJ, Stein GF, Kominsky JR, Ruhe RL, Watanabe AS: Common source community and industrial exposure to trichloroethylene. *Arch Environ Health* 42:327-332, 1987.
69. Rinsky RA, Smith AB, Hornung R, Filloon TG, Young RJ, Okun AH, Landrigan PJ: Benzene and Leukemia: An epidemiologic risk assessment. *New Engl J Med* 316:1044-1050, 1987.

70. Schwartz E, Landrigan PJ: Use of court records for supplementing occupational disease surveillance. *Am J Public Health* 77:1457-1458, 1987.
71. Rinsky RA, Melius JM, Hornung RW, Zumwalde RD, Waxweiler RJ, Landrigan PJ, Bierbaum PJ, Murray WE Jr: Case-control study of lung cancer in civilian employees at the Portsmouth Naval Shipyard, Kittery, Maine. *Am J Epidemiol* 127:55-64, 1988.
72. Schwartz J, Landrigan PJ, Feldman RG, Silbergeld EK, Baker EL Jr, VonLindern IH: Threshold effect in lead-induced peripheral neuropathy. *Pediatrics* 112:12-17, 1988.
73. Kraut A, Lilis R, Marcus M, Valciukas JA, Wolff MS, Landrigan PJ: Neurotoxic effects of solvent exposure on sewage treatment workers. *Arch Environ Health* 43:263-268, 1988.
74. Nicholson WJ, Landrigan PJ: Quantitative assessment of lives lost due to delay in regulation of occupational exposure to benzene. *Environ Health Perspect* 82:185-188, 1989.
75. Landrigan PJ, Halper LA, Silbergeld EK: Toxic air pollution across a state line: Implications for the siting of resource recovery facilities. *J Public Health Policy* 10:309-323, 1989.
76. Marino PE, Franzblau A, Lilis R, Landrigan PJ: Acute lead poisoning in construction workers - The failure of current protective standards. *Arch Environ Health* 44:140-145. 1989.
77. Fahs MC, Markowitz SB, Fischer E, Shapiro J, Landrigan PJ: The health costs of occupational disease in New York State. *Am J Ind Med* 16:437-449, 1989.
78. Landrigan PJ, Markowitz SB: Current magnitude of occupational disease in the United States: Estimates from New York State. *Ann NY Acad Sci* 572:27-45, 1989.
79. Wolff MS, Herbert R, Marcus M, Rivera M, Landrigan PJ, Andrews LR: PAH residues on skin in relation to air levels among roofers. *Arch Environ Health* 44:157-163, 1989.
80. Markowitz S, Landrigan P: The magnitude of the occupational disease problem: An investigation in New York State. *Toxicol Ind Health* 5:9-30, 1989.
81. Lilienfeld DE, Chan E, Ehland J, Godbold J, Landrigan PJ, Marsh G, Perl DP: Rising mortality from motoneuron disease in the U.S.A., 1962-1984. *Lancet* 1:710-713, 1989.
82. Elliott LJ, Halperin WE, Landrigan PJ: Perspectives on opportunities toward a hazard-free bioprocessing environment. In*: Bioprocessing Safety: Worker and Community Safety and Health Considerations*, ASTM STP 1051, WC Hyer Jr (ed). Philadelphia: American Society for Testing and Materials, pp 20-26, 1990.
83. Schwartz J, Landrigan PJ, Baker EL Jr, Orenstein WA, VonLindern IH: Lead-induced anemia: Dose-response relationships and evidence for a threshold. *Am J Public Health* 80:165-168, 1990.
84. Marino PE, Landrigan PJ, Graef J, Nussbaum A, Bayan G, Boch K, Boch S: A case report of lead paint poisoning during renovation of a Victorian farm house. *Am J Public Health* 80:1183-1185, 1990.
85. Herbert R, Marcus M, Wolff MS, Perera FP, Andrews L, Godbold JH, Rivera M, Stefanidis M, Quing Lu X, Landrigan PJ, Santella RM: Detection of DNA adducts in white blood cells of roofers by ^{32}P Postlabeling. In: *Complex Mixtures and Cancer Risk. Lyon: International Agency for Research on Cancer*. (Eds) H Vainio, M. Sorsa & AJ McMichael, pp 205-214, 1990.
86. Lilienfeld DE, Chan E, Ehland J, Godbold J, Landrigan PJ, Marsh G, Perl DP: Two decades of increasing mortality from Parkinson's disease among the nation's elderly. *Arch Neurol* 47:731-734, 1990.
87. Herbert R, Marcus M, Wolff MS, Perera FP, Andrews L, Godbold JH, Rivera M, Stefanidis M, Lu Q-X, Landrigan PJ, Santella RM: Detection of adducts of deoxyribonucleic acid in white blood cells of roofers by ^{32}P-postlabeling: Relationship of adduct levels to measures of polycyclic aromatic hydrocarbon exposure. *Scand J Work Environ Health* 16:135-143, 1990.

88. Lilienfeld DE, Chan E, Ehland J, Godbold J, Landrigan PJ, Marsh G: Mortality from pulmonary embolism in the United States: 1962-1984. *Chest* 98:1067-1072, 1990.

89. Kraut A, Chan E, Lioy PJ, Cohen FB, Goldstein BD, Landrigan PJ: Epidemiologic investigation of a cancer cluster in professional football players. *Environ Res* 56:131-143, 1991.

90. Rosenstock L, Rest KM, Benson JA Jr, Cannella JM, Cohen J, Cullen MR, Davidoff F, Landrigan PJ, Reynolds RC, Clever LH, Goldstein BD: Occupational and Environmental Medicine -- Meeting the Growing Need for Clinical Services (Special Article). *New Engl J Med* 325:924-927, 1991.

91. Johanning E, Wilder DG, Landrigan PJ, Pope MH: Whole-Body vibration exposure in subway cars and review of adverse health effects. *J Occup Med* 33:605-612, 1991.

92. Ehrlich R, Kattan M, Godbold J, Saltzberg DS, Grimm KT, Landrigan PJ, Lilienfeld DE: Childhood asthma and passive smoking: urinary cotinine as a biomarker of exposure. *Am Rev Respir Dis* 145:594-599, 1992.

93. Murata K, Landrigan PJ, Araki S: Effects of age, heart rate, gender, tobacco and alcohol ingestion on RR interval variability in human ECG. *J Autonomic Nervous System* 37:199-206, 1992.

94. Steenland K, Selevan S, Landrigan P: The mortality of lead smelter workers: an update. *Am J Public Health* 82:164l-1644, 1992.

95. McConnell R, Anderson K, Russell W, Anderson KE, Clapp R, Silbergeld EK, Landrigan PJ: Angiosarcoma, porphyria cutanea tarda and probable chloracne in a worker exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin-contaminated waste oil. *Br J Ind Med* 50:699-703, 1993.

96. Belville R, Pollack SH, Godbold JH, Landrigan PJ: Occupational injuries among working adolescents in New York State. *JAMA* 269:2754-2759 1993.

97. Markowitz SB, Nunez CM, Klitzman S, Munshi AA, Kim WS, Eisinger J, Landrigan PJ: Lead Poisoning Due to Hai Ge Fen. The porphyrin content of individual erythrocytes. *JAMA* 271:932-934, 1994.

98. Herbert R, Gerr F, Luo J, Harris-Abbott D, Landrigan PJ: Peripheral neurologic abnormalities among roofing workers: sentinel case and clinical screening. *Arch Environ Health* 50:349-354, 1995.

99. Lightdale JR, Oken E, Klein WM, Landrigan PJ, Welty TK: Psychosocial barriers to health promotion in a American Indian population. *American Indian and Alaska Native Mental Health Research* 7:34-49, 1997.

100. Herbert R, Plattus B, Kellogg L, Luo J, Marcus M, Mascolo A, Landrigan PJ: The union health center: A working model of clinical care linked to preventive occupational health services. *Am J Ind Med* 31:263-273, 1997.

101. Smith D, Herbert R, Schechter C, Phillips R, Diamond J, Carroll S, Weiner J, Dahms TE, Landrigan P: Occupational Coronary Heart Disease Among Bridge and Tunnel Officers. *Canadian J Cardiol* 13(Supp B):76B, 1997.

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INVITED LECTURES/PRESENTATIONS

Visiting Professorships and Lectureships:

University of Utah, Wallace Stegner Lecturer, 2012

Harvard School of Public Health, The James L. Whittenberger Lecturer, 2009

University of Kentucky, Inaugural John P. Wyatt Lecturer in Environmental Health and Disease, 2008

University of Minnesota, School of Public Health, Richard G. Bond Memorial Lecture, 2007

James P. Keogh, MD Memorial, Lecturer in Occupational Medicine, University of Maryland School of Medicine, 2006

Royal College of Physicians (London), Faculty of Occupational Medicine, Richard Schilling Memorial Lecturer, 2000

University of Rochester, 44th Annual Paul W. Beaven Lecturer, 2000

Centers for Disease Control and Prevention, Langmuir Memorial Lecturer, 1999

Mayo Clinic, Department of Pediatrics, Amberg-Helmholtz Lecturer in Pediatrics, 1998

Duke University Medical School, Visiting Professor, NIEHS Clinical Training Program in Environmental Medicine, 1995

National University of Singapore, Visiting External Examiner in Occupational Medicine, 1994

Medical College of Pennsylvania, Catherine Boucot Sturgis Visiting Professor in Community and Preventive Medicine, March 1992

University of Cape Town Medical School, Visiting Professor, Department of Community Health, March 1992

University of Tokyo, Visiting Professor of the University, July 1990

University of Tokyo, Visiting Professor of the Faculty of Medicine, September 1989

Exhibit 2

By Philip J. Landrigan and Lynn R. Goldman

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Children's Vulnerability To Toxic Chemicals: A Challenge And Opportunity To Strengthen Health And Environmental Policy

Philip J. Landrigan (phil.landrigan@mssm.edu) is the dean for global health and a professor of both preventive medicine and pediatrics at the Mount Sinai School of Medicine, in New York City.

Lynn R. Goldman is the dean of the School of Public Health and a professor of environmental and occupational health at the George Washington University, in Washington, D.C.

ABSTRACT **A key policy breakthrough occurred nearly twenty years ago with the discovery that children are far more sensitive than adults to toxic chemicals in the environment. This finding led to the recognition that chemical exposures early in life are significant and preventable causes of disease in children and adults. We review this knowledge and recommend a new policy to regulate industrial and consumer chemicals that will protect the health of children and all Americans, prevent disease, and reduce health care costs. The linchpins of a new US chemical policy will be: first, a legally mandated requirement to test the toxicity of chemicals already in commerce, prioritizing chemicals in the widest use, and incorporating new assessment technologies; second, a tiered approach to premarket evaluation of new chemicals; and third, epidemiologic monitoring and focused health studies of exposed populations.**

Recognition of the unique vulnerability of children, infants, and fetuses to toxic chemicals in the environment was a watershed development for health and environmental policy.[1] This discovery catalyzed two further insights: that early life exposures to toxic chemicals are important causes of disease and dysfunction in children and also in adults,[2–4] and that diseases caused by chemicals can successfully be prevented, thus saving lives, enhancing the quality of life, reducing health care and education costs, and increasing national productivity. A notable example is the nation's experience with removing lead from gasoline. This one change reduced lead poisoning by more than 90 percent[5] and produced an estimated annual economic benefit of $110 billion to $319 billion.[6]

These insights have affected risk assessment, regulation, and law.[7] In this article we explore the implications for health and environmental policy.

Children Are Vulnerable To Toxic Chemicals

The realization that children are uniquely sensitive to toxic chemicals was catalyzed by the publication in 1993 of a National Academies report, *Pesticides in the Diets of Infants and Children*.[1] Studies cited in the report found that children are quantitatively and qualitatively different from adults in their sensitivity to pesticides and other chemicals.

Prior to the report's publication, virtually all environmental policy in the United States had focused on assessment of risk to the "average adult." Risk assessment had paid scant heed to exposures that diverged from the norm. Little attention was paid to the unique risks of infants, children, or other vulnerable groups within the population.

The report produced a paradigm shift in that approach to health and environmental policy. It led to new legislative and regulatory initiatives to better protect infants and children against environmental health threats and has been especially

influential in changing the regulation of pesticide and pharmaceutical chemicals.[7]

The report identified four differences between children and adults that contribute to children's heightened susceptibility to chemicals in the environment.

First, children have greater exposures to toxic chemicals for their body weight than adults.[1] A six-month-old infant drinks seven times more water per pound than an adult.[8] Children take in three to four times more calories per pound than adults. The air intake per pound of an infant is twice that of an adult. These differences result in children being disproportionately exposed to toxic chemicals in air, food, and water. Children's hand-to-mouth behavior and play on the ground further magnify their exposures.

Second, children's metabolic pathways are immature,[1] and a child's ability to metabolize toxic chemicals is different from an adult's. In some instances, infants are at lower risk than adults because they cannot convert chemicals to their toxic forms. More commonly, however, children are more vulnerable because they lack the enzymes needed to break down and remove toxic chemicals from the body.[9]

Third, children's early developmental processes are easily disrupted.[1] Rapid, complex, and highly choreographed development takes place in prenatal life and in the first years after birth, continuing more slowly throughout childhood into puberty. In the brain, for example, billions of cells must form, move to their assigned positions, and establish trillions of precise interconnections.[10] Likewise, development of the reproductive organs is guided by a complex and precisely timed sequence of chemical messages and is shaped by maternal and fetal hormones.[11]

Recent research in pediatrics and developmental toxicology has elaborated the concept of "windows of vulnerability."[12] These are critical periods in early development when exposures to even minute doses of toxic chemicals—levels that would have no adverse effect on an adult—can disrupt organ formation and cause lifelong functional impairments.

If, for example, cells in an infant's brain are injured by lead or a pesticide, the consequences can include developmental disabilities in childhood[11,13] and possibly increased risk of neurological degeneration, such as Parkinson's disease, in adult life.[4] If inappropriate hormonal signals are sent to the developing reproductive organs by a synthetic chemical endocrine disruptor—such as certain chemicals commonly found in household products, plastics, and cosmetics (phthalates), and on clothing (flame retardants)—lifelong reproductive impairment may ensue.[11] These windows of vulnerability have no equivalent in adult life.

Fourth, children have more time than adults to develop chronic diseases. Many diseases triggered by toxic chemicals, such as cancer and neurodegenerative diseases, are now understood to evolve through multistage, multiyear processes that may be initiated by exposures in infancy.[1,4] This insight has catalyzed new research to identify how early environmental influences may affect health in childhood and across the human lifespan. Notable research includes the US National Children's Study,[14] the Japan Environment and Children's Study,[15] and the International Childhood Cancer Cohort Consortium.[16]

Rates Of Chronic Diseases In US Children Are Rising

Today in the United States, the principal causes of sickness, disability, and death in children are chronic illnesses. Rates of many of these diseases are high and rising.[2,3] Toxic chemicals in the environment are making important contributions to these disease trends.

Asthma is one of the most common chronic diseases in American children. The prevalence of childhood asthma has more than doubled over the past twenty years, and in 2008, 9 percent of all US children had asthma.[17,18] Asthma is the leading cause of pediatric hospitalization and school absenteeism and a major driver of pediatric health costs.

Birth defects are now the leading cause of infant death and are associated with substantial health and education costs. Certain birth defects, such as those of the male reproductive organs[19] and of the abdominal wall,[20] appear to have increased in frequency.

Neurodevelopmental disorders, including dyslexia, mental retardation, attention deficit hyperactivity disorder, and autism, affect 5–10 percent of the babies born in the United States each year.[21] Autism spectrum disorder is currently diagnosed in one of every 110 American children.[22] The prevalence of attention deficit hyperactivity disorder has also risen, and today 14 percent of US children have been diagnosed with this condition; two-thirds of them also have learning disabilities.[23]

The incidence of leukemia and brain cancer in children younger than age eighteen increased steadily from the 1970s through the 1990s, despite declining mortality.[24] Testicular cancer in males ages 15–30 has increased in incidence by more than 50 percent.[24]

Obesity in children has tripled in prevalence over the past twenty years, from 5 percent to

17 percent.[25] One of its consequences, type 2 diabetes, is occurring earlier in life and at epidemic rates.

Children And The Chemical Environment

The environment in which American children live has changed greatly in the past fifty years, especially in terms of the chemicals to which they are routinely exposed. During this time, more than 80,000 new synthetic chemicals have been invented and are used today in millions of consumer products, ranging from foods and food packaging to clothing, building materials, cleaning products, cosmetics, toys, and baby bottles.[26] Some of these chemicals may pose risks for children's health. The Environmental Protection Agency has identified 3,000 "high–production-volume" chemicals—chemicals produced in quantities of more than a million pounds per year—that are in widest use and therefore have the greatest potential for human exposure. Children are especially at risk for exposure to these chemicals.

In national surveys conducted by the Centers for Disease Control and Prevention, measurable quantities of 200 high–production-volume chemicals have been detected in the blood and urine of virtually all Americans,[27] including pregnant women.[28] The significance of this finding for human health is not fully understood. But it is worrisome, because most of these chemicals have not undergone even minimal assessment for potential toxicity, and only about 20 percent of them have been screened for their potential to disrupt early human development or to cause disease in infants and children.[26] Even less is known about the potential effects of exposure to several of these chemicals simultaneously, or how they may interact with one another in the human body, possibly causing synergistic adverse effects on health.

The absence of information about the possible risks associated with routine exposure to untested synthetic chemicals is fraught with risk for disease and dysfunction. Unless studies are conducted to specifically seek ill effects associated with chemical exposures, dysfunctions can go unrecognized for many years.

The "silent epidemic" of childhood lead poisoning[6,13] is a dramatic case in point. Millions of American children were exposed to excessive levels of lead from the 1940s to the 1970s, when lead was an additive to gasoline. Many suffered unrecognized brain injury before sufficient evidence could be marshaled to mandate removing lead from gasoline, household paint, and consumer products.[5,6]

Failure to evaluate chemicals for potential toxicity reflects the failure of the Toxic Substances Control Act of 1976.[29] At the time of its passage, the act was intended to be pioneering legislation that would require testing chemicals already in commerce for potential toxicity, and would also require premarket evaluation of all new chemicals. The act never fulfilled these intentions. A particularly egregious lapse was a decision by Congress to "grandfather in" 62,000 chemicals already on the market without any toxicity testing requirement.[29,30] These chemicals were presumed to be safe and allowed to remain in commerce, unless and until the Environmental Protection Agency made a finding that they posed an "unreasonable risk."[30]

The "unreasonable risk" standard identified in the Toxic Substances Control Act has created a substantial barrier to the regulation of industrial and consumer chemicals. This standard has been so burdensome that the Environmental Protection Agency has not been able to remove chemicals from the market except when there is overwhelming evidence of potential harm. The result is that only five chemicals have been controlled under the act in the thirty-five years since its passage. These chemicals were polychlorinated biphenyls (PCBs), chlorofluorocarbons, dioxin, asbestos, and hexavalent chromium. Only two of these five were totally banned: PCBs, which were eliminated by an act of Congress and not because the Environmental Protection Agency exercised its authority, and asbestos, a chemical for which there is overwhelming evidence of serious hazard to human health.

Further barriers to enforcement of the Toxic Substances Control Act have resulted from the federal courts' interpretation of the "unreasonable risk" standard. Thus, in a 1991 opinion on the asbestos ban in *Corrosion Proof Fittings v. EPA*, the Fifth Circuit found that the Environmental Protection Agency had failed to show that it was taking the "least burdensome" approach required under the act in formulating its final rule banning asbestos. The court thus overturned the agency's rule. This interpretation has made it virtually impossible since 1991 for the Environmental Protection Agency to regulate dangerous chemicals under the act.[30]

Toxic Chemicals And Disease In Children

Evidence is strong and continuing to accumulate that toxic chemicals are important causes of disease and dysfunction in children. This recognition first arose in studies of lead and mercury.[31–36] In recent years, as research strategies in environmental pediatrics have become more refined, the

pace of scientific discovery has quickened and a series of new associations has been discovered. Examples include the following.

Prenatal exposure to PCBs is associated with reduction in children's intelligence.[37] PCBs are an environmentally persistent class of chemicals that accumulate to high levels in certain species of fish. Human exposure is principally the consequence of maternal consumption of contaminated fish before and during pregnancy. Although PCBs are no longer manufactured in the United States, they were used extensively for many years in manufacturing electrical equipment such as transformers, and they continue to be important contaminants today because they are highly persistent in the environment and because they become concentrated in the tissues of organisms in the food chain.

Prenatal exposure to the commonly used insecticide chlorpyrifos is associated with reduced head circumference at birth [38] and with developmental delays.[39] Small head circumference at birth is an indicator of delayed brain growth during pregnancy. Chlorpyrifos is also linked to pervasive developmental disorder, a form of autism.[39,40]

Baby boys exposed in the womb to phthalates—a chemical compound found in plastics, cosmetics, and many common household products—appear to be at increased risk of behavioral abnormalities that resemble attention deficit hyperactivity disorder.[41] Prenatal exposure to bisphenol A, a synthetic chemical used to manufacture polycarbonate plastics, is linked to behavioral abnormalities in girls.[42] Prenatal exposure to brominated flame retardants is linked to cognitive impairments,[43] and prenatal exposures to arsenic and manganese is associated with neurodevelopmental impairment.[44,45]

Rates of asthma are increased in children exposed to secondhand cigarette smoke and to fine particulate air pollution.[17,18] Risk of respiratory death is increased in infants exposed to fine particulate air pollution.[46]

Prenatal exposure to phthalates has also been linked to shortening of the ano-genital distance in baby boys, a finding indicative of feminization.[47] Prenatal exposure to perfluorinated chemicals (perfluorooctanic acid and perfluorooctane sulfonate) used to make nonstick pans and stain repellents has been linked to decreased birthweight and reduced head circumference in newborn infants.[48]

Diseases Associated With Chemicals Are Costly

Preventing exposures to chemicals can yield great savings. To estimate the contribution of environmental pollutants to the prevalence and costs of disease in American children, investigators at Mount Sinai School of Medicine examined four categories of illness: lead poisoning, asthma, cancer, and neurobehavioral disorders.[49] Based on prevalence, the environmentally attributable fraction of each disease, and national economic data, they calculated that the total annual costs of these diseases attributable to environmental exposures is $54.9 billion (range $48.8 billion to $64.8 billion): $43.4 billion for lead poisoning, $2.0 billion for asthma, $0.3 billion for childhood cancer, and $9.2 billion for neurobehavioral disorders. Because of the difficulties inherent in assessing the full economic consequences of neurobehavioral impairments, it is likely that these estimates are low.

Disease and dysfunction caused by toxic chemicals can be prevented. Prevention is most effectively achieved by assessing chemicals for toxicity through laboratory and human studies and using the data gained in those assessments to guide evidence-based prevention of exposure. Great cost savings can result.

Again, we use the example of phasing out the use of lead in gasoline. This phase-out began in the United States in 1976, was 50 percent accomplished by 1980, and virtually complete by 2000.[5] Prior to 1976, 100,000 tons of tetraethyl lead was added to the US gasoline supply each year to improve engine performance and fuel efficiency. Widespread environmental contamination resulted.

The average US blood lead level peaked in the mid-1970s at 17 micrograms per deciliter,[5] a level significantly above the current Centers for Disease Control and Prevention guideline of 10 micrograms per deciliter and now known to be associated with significant toxic injury to the developing brain. These elevated blood lead levels, found in epidemiologic studies, were associated with reduced intelligence, shortened attention span, and disruptive behavior in children.[33,34] Each increase of 3 micrograms per deciliter in mean blood lead level was shown to be associated with a decline of 0.5–1.0 points in intelligence quotient (IQ).[6] These effects occurred in the absence of any clinical symptoms or obvious illness and were thus termed "silent" lead poisoning.[34]

The discovery that lead could erode children's intelligence even at relatively low levels was not the original justification for the Environmental Protection Agency's decision to remove lead from gasoline. In fact, the decision to remove lead was made in the first instance to protect catalytic converters from damage by lead. However, the discovery did play an important role in reinforcing the decision and in sustaining it over

time. A result of the phase-out was that between 1976 and 1990 the mean blood lead level of American children decreased by more than 90 percent (to below 2 micrograms per deciliter today).[5] The incidence of childhood lead poisoning also fell by more than 90 percent.[5]

A further consequence of the reduction in exposure to lead was that the mean IQ of American children has increased.[6] Children born in the United States today are estimated to have IQ scores that, on average, are 2.2–4.7 points higher than those of children born in the early 1970s.[6] And because each 1-point gain in population mean IQ is associated with an estimated 2 percent increase in productivity over a lifetime,[50] the gain in population IQ is estimated to have produced a national economic benefit of $110–$319 billion in each annual cohort of babies born in the United States since the 1980s.[6]

Consequences For Environmental Policy

The recognition of children's unique vulnerability to toxic chemicals has had far-reaching consequences.

LEGISLATIVE CONSEQUENCES Recognition of children's susceptibility to toxic chemicals strongly influenced the Food Quality Protection Act of 1996, the major federal law governing the use of pesticides. This act became the first federal environmental statute to contain explicit provisions for protecting children's health.

This recognition led also to passage of the Best Pharmaceuticals for Children Act of 2002. This act requires that drugs labeled for use in children undergo studies to specifically examine children's susceptibilities.

CONSEQUENCES FOR RISK ASSESSMENT AND REGULATION A key provision of the Food Quality Protection Act is a requirement that federal pesticide standards ("tolerances") be health-based and that they explicitly consider the effects of pesticides on children's health.[30,51] This requirement represents a diametric change from the previous regulatory regime, in which the health risks of pesticides were balanced against the costs of regulation to agricultural producers in setting standards. This provision of the act forced reexamination of all extant pesticide tolerances to ensure that they met the standard of public health protection. As a result, many uses of pesticides were reduced or dropped altogether.

For example, agricultural use of organophosphate insecticides, a class of pesticide chemicals toxic to brain development, was reduced.[52] The review led also to bans on residential applications of two widely used insecticides—chlorpyrifos and diazinon—that had been used for household pest control.[52]

The Food Quality Protection Act mandates realistic consideration of exposures to multiple pesticides via multiple routes to assess potentially synergistic effects.[53] The law also mandates consideration of exposures to pesticide chemicals that are endocrine disruptors. These are chemicals that exert their toxicity through interactions with the endocrine system, disrupting function of the thyroid or pituitary glands, the ovaries, or the testes, or changing levels of hormones by changing their metabolism.[11]

The new approaches to risk assessment mandated by the Food Quality Protection Act have not yet extended beyond pesticides to include industrial or consumer chemicals.

CONSEQUENCES FOR BIOMEDICAL RESEARCH Recognition of children's vulnerability led to establishment of the Office of Children's Health Protection within the Environmental Protection Agency.[7] It catalyzed a 1997 executive order requiring federal agencies to consider children's special susceptibilities in all policy and rule making.[54] And it led to the creation of a White House Task Force on Children's Health and Safety.

Those programs have, in turn, stimulated substantial investments in children's health research.[7] The resulting initiatives include the following: a national network of Centers for Children's Environmental Health and Disease Prevention Research, supported by the National Institute of Environmental Health Sciences and the Environmental Protection Agency;[55] a network of Pediatric Environmental Health Specialty Units supported by the Centers for Disease Control and Prevention and the Agency for Toxic Substance and Disease Registry;[56] fellowship training programs in environmental pediatrics;[57] and the National Children's Study, a prospective epidemiologic study that will follow a nationally representative sample of 100,000 children from early pregnancy to age twenty-one.[14]

Consequences For Health Policy

The finding that children are uniquely vulnerable to synthetic chemicals indicates the need for fundamental revision of US chemical policy. By default, current policy presumes chemicals to be safe and permits them to enter and remain on the market with minimal evaluation of potential toxicity unless and until they are proved to be overwhelmingly hazardous by the Environmental Protection Agency, using the Toxic Substances Control Act's standard of "unreasonable risk." This policy is neither protective of human health nor consistent with current scientific

understanding of children's vulnerability.

The credible possibility exists that among the hundreds of untested chemicals currently in wide commercial use, there are synthetic chemicals whose toxicity to early childhood development has not yet been discovered.[13] The late David Rall, former director of the National Institute of Environmental Health Sciences, once stated, "If thalidomide [a drug widely used in the 1950s and 1960s to treat morning sickness in early pregnancy] had caused a ten-point loss of IQ instead of obvious birth defects of the limbs, it would probably still be on the market."[58]

To protect human health, and especially the health of infants and children, the paradigm for regulating industrial and consumer chemicals needs to become health-based. The Toxic Substances Control Act's "unreasonable risk" standard needs to be replaced with a new standard that explicitly considers effects of industrial and consumer chemicals on children's health. Such a move would align the strategy used to regulate industrial chemicals with the strategy used under the Food Quality Protection Act to regulate pesticides. It would mark a dramatic change in the current regulatory regime.[30]

A New US Chemical Policy

The linchpin of a new, health-based chemical policy would be a legally mandated requirement that chemicals already on the market be systematically examined for potential toxicity. Such testing will not be an easy task, but it is necessary. It will be far more challenging than updating the tolerances for pesticides proved to be.

To evaluate tens of thousands of chemicals currently in commercial use would require new legislation that directed the Environmental Protection Agency to first address those classes of chemicals that are in the widest use and the most likely to confer risk. Data on the use of chemicals in consumer products, especially products used by young children and pregnant women; data on discharges of chemicals into the air and water; and data on chemicals already widely detectable in the bodies of Americans[27] would help to target the chemicals that most urgently need to be evaluated.[30]

Enhanced evaluation of chemical toxicity would require new, more efficient approaches to toxicity testing. Such approaches are already in development at the National Institute of Environmental Health Sciences and the Environmental Protection Agency.[59] These approaches should incorporate new technologies identified through research in developmental toxicology and consider such complexities as which endpoints to assess, which doses to administer, and which mixtures to evaluate.[59]

A second critical component of a health-based chemical policy would be a legally mandated, strictly enforced requirement that all new chemicals be assessed for potential toxicity before they enter the market. Such assessment could be undertaken in tiered fashion, making use of new rapid assessment methods in computational and in vitro toxicology, taking into account the proposed use patterns of new chemicals, and giving the Environmental Protection Agency latitude to require less extensive evaluation of chemicals and chemical uses judged to be less hazardous to health.[30,59]

As has happened with pesticides, the new approach to the evaluation of industrial chemicals that we propose here would be more likely to result in continued approval for certain uses and withdrawal of approval for others, rather than outright bans of chemicals. For example, the United States, Canada, and the European Union have all recently taken action to ban polycarbonate plastics containing bisphenol A from baby bottles. However, in all of these regions, polycarbonates are still permitted in the manufacture of compact discs, eyeglasses, and other consumer products in which the potential for human exposure is judged to be lower than in uses where the bisphenol A can migrate into foods.

One model approach to health-based chemical policy can be found in the European Union's Registration, Evaluation, Authorisation and Restriction of Chemical Substances legislation, enacted in 2007.[60] This legislation, commonly referred to by its acronym, REACH, places the responsibility on industry to generate substantial amounts of data on potential risks of commercial chemicals and to register this information in a central database that is housed in the European Chemical Agency in Helsinki.[61] The European Chemical Agency not only manages this central database but also coordinates the in-depth evaluation of suspicious chemicals. It is also developing a public database to house and make accessible hazard information relevant to consumers and health and environmental professionals. The first cycle of REACH registrations closed in January 2010 and in February 2011 the European Chemical Agency released its first list of six dangerous substances that are to be phased out by the European Union, through a process that involves scientific analysis and consultation with member states. The European Union is using this information to craft regulations that protect the health of European children, and it has led to bans and restrictions of certain potentially toxic products.[61]

Much of the information collected by the Euro-

pean Union under REACH is claimed as confidential business information and is therefore not available to the US government or to any other entities outside of European Union regulatory authorities. A new, health-based US chemical policy could mandate that industry provide similar data to US regulators. Because these data are already being produced for use in Europe, the marginal costs of providing them to the Environmental Protection Agency should not be great.

A third pillar of a health-based chemical policy would be continued research to examine the impact of chemicals on children's health.[14,55,56] Such research, which includes epidemiologic monitoring of exposed populations as well as specific studies of the effects of particular chemicals, is an essential complement to toxicity testing. It provides direct evidence of the effects of chemicals on human health. It also provides an evidentiary basis for assessing the impact on children's health of policy interventions.

The argument will probably be made that any additional controls on chemicals would cost jobs and harm the economy. However, there is little evidence that environmental protection has to date been harmful to the US economy or to business.[62] To the contrary, there is compelling evidence that the high costs of diseases caused by toxic chemicals are a major, but potentially avoidable, drag on the US economy.[49,63]

Conclusion

Recognition of children's unique vulnerability to toxic chemicals, a vulnerability that receives scant consideration in current US chemical policy, challenges existing policy and creates an opportunity for change.

Creating a new chemical policy explicitly protective of health could prevent disease and dysfunction in childhood and across the lifespan, reduce health and education costs, increase national productivity, and promote better health and well-being for all Americans. ■

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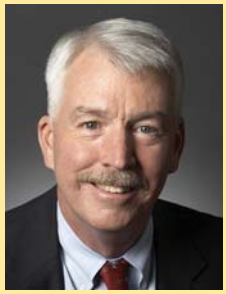
ABOUT THE AUTHORS: PHILIP J. LANDRIGAN & LYNN R. GOLDMAN



Philip J. Landrigan is the dean for global health and a professor of both preventive medicine and pediatrics at the Mount Sinai School of Medicine.

In *Health Affairs* this month, Philip Landrigan and Lynn Goldman review findings that children are far more sensitive to environmental toxins than are adults—and on that basis, the authors argue for an overhaul of our system for regulating chemicals. They call for a requirement that all chemicals to be introduced into the market, as well as those already on the market, be tested for toxicity. What's more, they argue that chemicals' actual or potential impact on all exposed populations, including children, should be taken into account in the testing and review process.

Landrigan, who is also the subject of a "People and Places" article in this issue of *Health Affairs*, is an epidemiologist and pediatrician, and the Ethel Wise Professor of preventive medicine at the Mount Sinai School of Medicine. He is also the school's dean for global health and the director of the Children's Environmental Health Center. He has long been associated with research demonstrating that children are more susceptible than adults to environmental exposures, such as to lead and pesticides.

Landrigan is a recipient of the Meritorious Service Medal of the Public Health Service and a member of the Institute of Medicine. He received his medical degree from Harvard Medical School and a master of science degree in occupational medicine from the University of London.

Lynn R. Goldman is the dean of the School of Public Health and a professor of environmental and occupational health at the George Washington University.

Goldman is the dean of the George Washington University School of Public Health. Previously, she was a professor of environmental health sciences at the Johns Hopkins University's Bloomberg School of Public Health. Before that, she served in the Clinton administration, as assistant administrator for toxic substances in the Environmental Protection Agency. During her time there, the agency overhauled the nation's pesticide laws, expanded right-to-know requirements for release of toxins, reached consensus on an approach to testing chemicals with the potential to disrupt the human endocrine system, developed standards to implement lead screening legislation, and promoted children's health and global chemical safety.

Goldman also worked in environmental health for the California Department of Public Health Services, where she managed a statewide environmental epidemiology program that focused on childhood lead poisoning, birth defects, and occupational health. She is a member of the Institute of Medicine and of the National Academy of Sciences Board on Environmental Sciences and Toxicology.

Goldman earned a master's degree in health and medical science from the University of California, Berkeley; a master of public health degree in epidemiology from the Johns Hopkins University School of Hygiene and Public Health; and a medical degree from the University of California, San Francisco.